



Aged spontaneously hypertensive rats (SHR) have increased oxidative stress as measured by high levels of oxidized low-density lipoprotein (oxLDL) that can activate plasminogen activator inhibitor (PAI-1) through NADPH oxidase. Increased PAI-1 could increase amyloid-beta42 (Aβ42) deposition through increased protease inhibition. During middle cerebral artery occlusion (MCAO), impaired collateral perfusion in both young and aged SHR was increased by PAI-1 inhibition (TM5441) in a nitric oxide (NO)-dependent manner that likely contributed to the decrease in infarction. However, PAI-1 inhibition did not affect hemorrhage or erythrocyte aggregation in aged SHR.